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The Effects of Respiratory Sinus Arrhythmia Biofeedback on Heart Rate Variability and Posttraumatic Stress Disorder Symptoms: A Pilot Study

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Abstract Recent studies have found a significant association between PTSD and low heart rate variability (HRV), a biomarker of autonomic dysregulation. Research indicates that respiratory sinus arrhythmia (RSA) biofeedback increases HRV while reducing related pathological symptoms. This controlled pilot study compared RSA biofeedback to progressive muscle relaxation (PMR) as adjunctive interventions for 38 persons with PTSD symptoms in a residential treatment facility for a substance use disorder. Both groups were assessed at pre-intervention and 4-week post-intervention. Group × time interactions revealed significantly greater reductions in depressive symptoms and increases in HRV indices for the RSA group. Both groups significantly reduced PTSD and insomnia symptoms and a statistical trend was observed for reduced substance craving for the RSA group. Increases in HRV were significantly associated with PTSD symptom reduction. Overall, these results provide preliminary support for the efficacy of RSA biofeedback in improving physiological and psychological health for individuals with PTSD.

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Introduction

Posttraumatic stress disorder (PTSD) is a prevalent, often chronic, psychiatric disorder characterized by an intense response of fear, horror, or helplessness to trauma exposure. Its symptoms include intrusive reexperiencing and maladaptive avoidance of the focal trauma as well as hyperarousal symptoms (American Psychiatric Association 2000). Research indicates that heightened physiological activity, or hyperarousal, predicts PTSD onset and symptom severity (Breslau and Kessler 2001; Bryant 2005; Elsesser et al. 2005; Schell et al. 2004).

Elevated heart rate (HR) is the most prominent autonomic feature of PTSD, and its chronicity may be a risk factor for cardiac-related pathologies (Blanchard 1990; Buckley and Kaloupek 2001). A multi-site study found that elevated HR elicited by trauma cues could correctly classify PTSD and non-PTSD subjects for two-thirds of the sample (Keane et al. 1998). Similarly, a meta-analysis of neuroimaging studies identified two neurophysiological responsivity patterns provoked by trauma cues: an arousal subtype with approximately 70% of individuals having elevated HR and a dissociative one with 30% having no HR increase (Lanius et al. 2006). Additionally, a recent study revealed that elevated HR coupled with increased respiratory rate may predict the onset of PTSD (Bryant et al. 2008).

A biomarker of autonomic nervous system (ANS) functioning is heart rate variability (HRV). Similar to several other visceral organs the heart is dually innervated

by both the parasympathetic and sympathetic branches of the ANS. Fluctuations between consecutive R-to-R intervals provide a "dynamic map" of the interrelationship between the two branches of the ANS.

More recent studies have examined the relationship between PTSD and HR using HRV indices, a more precise physiological measure of HR functioning. Researchers hypothesize that higher amplitude HRV promotes autonomic homeostasis and the ability to self-regulate emotionally (Porges et al. 1994). Psychiatric and physiological disorders comorbid with PTSD, including depression (Stein et al. 2000) and insomnia (Bonnet and Arand 1998), are also associated with low HRV. Research with clinically depressed individuals indicates that respiratory sinus arrhythmia (RSA) biofeedback training facilitates an increase in HRV amplitude and a decrease in depressive symptoms (Karavidas et al. 2007). RSA biofeedback training involves pacing breath rhythms at approximately six breaths-per-minute so that a resonance between respiratory and baroreflex rhythms can occur, increasing overall HRV amplitude (Lehrer 2003; Lehrer et al. 2000).

Assessment studies using symptom provocation cue protocols indicate that both lower baseline and elicited RSA amplitude are frequently associated with a PTSD diagnosis as well as symptom severity. Two studies found that participants with PTSD compared to normal controls had lower RSA amplitudes at all three conditions–baseline, symptom provocation cue, and recovery (Cohen et al. 1997, 1998). However, a similar study found that RSA amplitude was only lower for a PTSD group during the trauma cue condition but not at baseline (Sahar et al. 2001). The mixed results for baseline RSA amplitude may be clarified by two studies that examined PTSD symptom severity. Participants with a PTSD diagnosis were stratified into symptom severity groups based on RSA amplitude and HR responses following a trauma-related script-driven imagery task. Only those participants with the lowest provoked HRV amplitudes had accompanying HR elevations (Hopper et al. 2006) and a more prolonged HR arousal and recovery period (Sack et al. 2004). This suggests that more severe autonomic dysregulation may influence elevated HR as well as chronic PTSD.

Increases in HRV have been associated with decreases in PTSD symptoms in a few pilot intervention studies. The treatments range from hatha yoga (van der Kolk 2006), fluoxetine treatment (Cohen et al. 2000); eye movement desensitization and reprocessing (EMDR; Sack et al. 2003); and cognitive behavioral therapy (CBT; Nishith et al. 2003). However, the efficacy of an RSA biofeedback intervention for the treatment of PTSD symptoms, and its effects on HRV, has not been investigated in a controlled study.

The purpose of this controlled pilot study was to compare the efficacy of a portable RSA biofeedback device to a guided progressive muscle relaxation (PMR) audio exercise on the symptoms of PTSD, depression, insomnia, and autonomic functioning in a sample of persons with elevated PTSD symptoms with comorbid substance use disorder (SUDs). Both interventions were given as adjuncts to the usual treatment participants received while in a residential facility for SUDs. It was hypothesized that individuals in the RSA biofeedback group would report greater reductions in PTSD and depressive symptoms and greater increases in HRV than those in the PMR condition. In addition, it was hypothesized that an increase in HRV would be associated with psychological symptom improvement. Exploratory hypotheses investigated whether RSA biofeedback would be associated with greater reductions in insomnia and substance craving.

Method

Participants

Participants were recruited from an urban residential therapeutic community program for the treatment of SUDs. The study was approved by the Alliant International University Institutional Review Board, as well as the research committee of the residential facility. The 6-month to 1-year residential treatment program provided psychosocial and psychotherapeutic treatment services. Participant selection was designed to include persons who endorsed one or more exposures to a traumatic event, had elevated PTSD symptoms, and were able to comply with the treatment intervention. Interested participants were eligible if they met the following criteria: (1) age range from 18 to 60, (2) minimum of 2 weeks abstinence from substance use behavior, and (3) elevated PTSD symptomology. Potential participants were excluded if they were unable to comply with study procedures due to past or present organic illnesses; expulsion or departure from the facility; or difficulty in obtaining a pulse reading from the RSA biofeedback device (attributed to thin fingers, low finger temperature, or poor circulation).

Seventy-six volunteers were screened for eligibility with the demographics questionnaire and the Posttraumatic Stress-Total (PTS-T) scale of the DAPS (Detailed Assessment of Posttraumatic States; Briere 2001). Twenty-three participants were excluded before randomization. Two declined to participate in the study and 21 participants did not meet eligibility criteria. Fifty-three participants received *T* scores of 60 and greater (considered above the "mild" PTSD symptom range for a trauma-exposed population). Initially, participants were randomly assigned to groups. Because the sample size was small, a stratification plan was created as the study progressed to balance groups on PTSD

symptom severity level, gender, and medication intake. To preserve an unbiased selection process, only the participant ID number and matching variables were included in the stratification process and then based on scores, participants were assigned into either the RSA biofeedback or PMR groups. This was achieved by using a stratification chart.

Of the eligible 53 participants, three were unable to register a finger pulse and were excluded, seven left the treatment facility after randomization but before beginning treatment, and five were excluded after refusing facility treatment services during the study. There were no significant differences between groups in terms of attrition. The final sample used in analyses was 38 participants, 19 in each group, who completed both pre-intervention to post-intervention assessments.

Measures

Each participant completed a demographic, a post-experimental, and psychometric questionnaires as well as a physiological assessment. The demographic questionnaire was a 14-item inventory that queried respondents on personal as well as medical characteristics. In addition, one question assessing substance craving on a 10-point scale from 1 (*no craving*) to 10 (*extreme craving*) scale was used as an exploratory outcome measure. The substance craving question was repeated on the 8-item post-experimental questionnaire, which was administered after the 4-week intervention period. All other psychometric questionnaires were reliable, valid, and widely used. The PTS-T scale from the DAPS (Briere 2001) and the PTSD Checklist-Civilian Version (PCL-C; Weathers et al. 1994) examined severity levels of PTSD symptoms; the Beck Depression Inventory II (BDI-II; Becket al. 1996) assessed levels of depressive symptoms; and the Insomnia Severity Index (ISI; Morin 1993) assessed insomnia symptom severity. The physiological assessment measured HRV amplitude using the standard deviation of the normal-to-normal beats (SDNN), a primary measure of HRV because it reflects the oscillating influences of the sympathetic and parasympathetic systems on the cardiac one.

Treatment Intervention Devices

Participants were assigned to use either an RSA biofeedback device, the StressEraser (Helicor, New York), or a 20-min PMR recording. Participants were provided with one of the two coded devices (the StressEraser or a compact disc player) for their personal use during the 4-week intervention period, were discretely instructed on how to use each specific one, and were asked to employ the devices daily for 20 min. Participants reported time and amount of use weekly on written logs. In addition, the StressEraser records the frequency of usage as well as the ratio of the number of points scored and length of time taken to achieve them. Both data points were used to examine the relationship between amount and efficiency of device usage and other continuous outcome measures.

The RSA biofeedback device is called the StressEraser, which is a class II (510(k) premarket notification exempt medical device that is indicated for relaxation training and stress reduction. The non-invasive, portable device assesses the RSA of each HR oscillations via an infrared finger photoplethysmograph. Users can achieve an optimal HR rhythm when breathing is paced at approximately six breaths-per-minute.

For the PMR group, a 20-min PMR exercise was derived from a track from the compact disc recording *Putting Your Worries on a Shelf* (*Long Progressive Muscle Relaxation*, Linehan 2005) and was recorded onto a compact disc. PMR was designated as the alternate intervention because muscle relaxation training has been found to be less effective than comparison treatments and is not empirically supported as a primary treatment in PTSD intervention studies (Foa et al. 2000a).

Psychophysiological Assessment

Physiological responses of the ANS were measured with a noninvasive biofeedback system using an I-330 C-2 interface (J & J Engineering, Poulsbo, WA) with Sony VAIO Core Duo laptop computer. Subjects were seated in front of the computer in a temperature-controlled room. Two electrocardiogram sensors, one on each wrist, were secured under sports wristbands; three sensors–two galvanic skin conductance sensors, one of which served as a ground, and a thermistor sensor–were attached to the fingertips on the non-dominant hand; and a respiration-monitoring belt with sensors was placed around the upper abdomen.

A 20-min physiological assessment session was administered individually at pre-intervention and post-intervention. Sessions were divided into three consecutive periods: baseline (*BASE*; 5 min of relaxing while listening to a non-arousing audio recording); paced breathing exercise (*RELAX*; 10 min of paced breathing); and recovery (*REC*; 5 min relaxing while listening to a non-arousing audio recording). During RELAX, participants were instructed to gaze at the computer screen and match the rhythm of their breathing to a slowly oscillating small sphere, set at six breaths-per-minute, as it moved across the screen. The RELAX procedure is a standardized office-based protocol for increasing HRV amplitude (Lehrer et al. 2000). To examine the influence of respiration on the SDNN measure, breaths-per-minute (BPM) was averaged across 60-s intervals by group for all three periods (J&J Engineering).

Data Reduction

Heart rate data were averaged across 60-s intervals at a sampling rate of 512 hertz and edited by averaging premature ectopic beats that exceeded a 25% difference between two consecutive data points (Task Force 1996). An HRV Analysis program (The Biosignal Analysis Group, Department of Physics, University of Kuopio, Finland) interpreted the standard deviation of all normal-to-normal RR intervals (SDNN; intervals between adjacent QRS complexes resulting from sinus node depolarizations). SDNN assessed the autonomic effects (HRV) of the two interventions.

Experimental Procedures

Residents were informed of the study parameters during group meetings, by informational fliers, or through case manager referrals. Participants were blind to the study hypotheses: they were told that the 4-week study would compare the relative effectiveness of muscle and breathing relaxation for persons with symptoms of trauma; and that it was unknown which would reduce symptoms, or if both or neither would help. Potential participants signed the informed consent form and then were screened for eligibility in a private room at the study site. Eligible participants completed baseline self-report questionnaires and were randomized or matched into their respective group. Within 1 week, each participant completed the psychophysiological assessment protocol and then received a 30-min training on their assigned device. The study was conducted over a period of approximately 3 months. All participants were debriefed upon study completion but before data analysis individually by the investigator. Participants still residing in the treatment were debriefed individually with a script; every effort was made to contact individuals who had left treatment with a letter. Both standardized forms discussed why participants were chosen for the study, the nature and treatment of PTSD, and the study hypothesis. Participants were encouraged to ask questions; and were given a research cell phone number for further questions and to learn about the outcome of the data analysis.

Statistical Analysis

Repeated measures analyses of variance (*RM ANOVAs*) were conducted to test the differential efficacy of the study treatments for decreasing PTSD, depressive, insomnia, and substance craving symptoms as well as for increasing SDNN. Chi square analyses were carried out to examine the changes in SDNN amplitude from pre-intervention to post-intervention. Three statistical outliers were removed from the SDNN analyses at baseline, two from the RSA group and one from the PMR group using the SPSS 14.0 explore function. Hierarchical multiple regressions were performed to examine the relationship of SDNN changes to changes in PTSD and related symptoms. Due to the exploratory nature of the data, no corrections for experiment wise error were used. Caution is therefore warranted for significant and trend level p-values with low effect sizes.

Results

The effectiveness of random assignment in achieving pretest group equivalence was examined by conducting t-tests and Chi square analyses. There were no significant differences between groups on any demographic or baseline variables, including medications (all $ps > .05$). Table 1 provides the demographic information of the participants.

PTSD Symptoms

There was no significant group × time interactions on either the PTS-T or the PCL-C. Within-group analyses using paired t-tests showed that both the RSA and PMR groups significantly reduced PTSD symptomology from pre-intervention to post-intervention on the PTS-T and the

Table 1 Demographic characteristics ($n = 38$)

Characteristic	*n*	%
Age		
18–30	7	18.4
31–40	12	31.6
41–50	13	34.2
51–60	6	15.8
Gender		
Male	21	55.3
Female	17	44.7
Ethnicity		
African American	21	55.3
White/Caucasian	12	31.6
Asian	2	5.3
Other	2	5.3
Hispanic/Latino	1	2.6
Education		
No high school	10	26.3
Graduate high school/GED some	13	34.2
College	13	34.2
College graduate	2	5.3

All participants were enrolled in a residential substance abuse treatment facility during the study

PCL-C (all $ps < .01$). Table 2 provides results of the repeated measures ANOVAs for the self-report and physiological assessment measures.

Comorbid Symptoms

As shown in Fig. 1, there was a significant group × time interaction effect for depression on the BDI-II, $F(1, 34) = 9.39$, $p < .01$. Within-group analysis using paired *t*-tests for dependent samples revealed that, as opposed to the PMR group, $t(35) = .034$, $p = .973$, the RSA group reduced depressive symptoms significantly, $t(35) = -2.15$, $p = .038$. Using the BDI-II categorical scores (0–13 = minimal depression; 14–19 = mild depression; 20–28 = moderate depression; 29–63 = severe depression), 94.1% of participants in the RSA group fell one category as compared to 57.9% of the PMR group, $\chi^2 = 6.28$, $p < .05$. Additionally, when removing participants who were in the minimal depression category at baseline ($n = 3$), 58.8% of participants in the RSA group were in the minimal depression category compared to 18.8% of the PMR group, $\chi^2 = 6.10$, $p < .05$ at end of treatment. There were no differences between groups on ISI scores and within-group analyses revealed that both groups significantly reduced ISI scores (all *p* values < .05). Similarly, no between group differences existed on substance craving but within-group analyses revealed reduced substance craving at a trend level ($p < .1$) in the RSA biofeedback group, $F(1, 15) = 3.39$, $p < .094$.

HRV Assessment

The baseline within-session paced breathing period (*Relax*) was examined to determine whether persons with PTSD symptoms were physiologically capable of increasing their HRV amplitude as well as to assess if the peak valley amplitudes of the two groups were comparable. Participants in both groups were able to increase SDNN values to just above .050 ms, an indicator of normal HRV for healthy adults (Task Force 1996). Independent *t*-tests were conducted at *Relax* and no significant difference was found between the two groups, $p > .05$.

Group × time repeated measures analyses of variance (RM ANOVAs) were then conducted on pre-intervention and post-intervention SDNN scores separately at all three periods of the HRV assessment protocol. There was a significant interaction effect for group × time for SDNN at BASE, $F(1, 33) = 5.81$, $p < .02$. As opposed to the PMR group, $t(17) = .58$, $p = .57$, the RSA group significantly increased SDNN at BASE, $t(15) = -2.4$, $p = .03$. A trend for a significant between-group effect was present for SDNN at REC, $F(1,33) = 3.94$, $p < .06$, with a significant within-group effect present for the RSA group only $F(1, 15) = 8.14$, $p < .02$ during REC. No significant between or within-group differences existed on SDNN at RELAX. Figure 2 provides SDNN values from pre-intervention to post-intervention by group and assessment period. As shown in Table 2, the RSA group had significant reductions in resting BPM at both BASE and REC compared to

Table 2 Between group differences, means and standard deviations for self-report and physiological assessments

Measure	RSA *M* (SD)		PMR *M* (SD)		*F*	*df*[a]	*p*
	Pre-intervention	Post-intervention	Pre-intervention	Post-intervention			
PTS-T	88.00 (12.57)	71.78 (15.41)	87.84 (11.74)	73.63 (16.86)	.12	36	.73
PCL-C	52.59 (11.86)	38.53 (10.03)	53.84 (10.18)	44.32 (13.59)	1.03	35	.32
BDI-II	26.35 (8.04)	12.29 (7.97)	25.95 (11.14)	19.47 (11.75)	6.78	35	.001
ISI	10.69 (06.58)	6.38 (5.35)	13.26 (5.76)	7.58 (5.05	.43	35	.52
CRAVE	4.13 (3.69)	2.44 (1.79)	3.11 (2.56)	3.16 (2.24)	2.52	34	.12
SDNN BASE	0.037 (.021)	0.054 (.037)	0.038 (.019)	0.036 (.016)	4.24	32	.048
SDNN RELAX	0.058 (.029)	0.060 (.030)	0.052 (.025)	0.048 (.023)	.69	32	.41
SDNN REC	0.046 (.019)	0.060 (.031)	0.045 (.021)	0.044 (.019)	3.94	32	.056
BPM BASE	15.01 (4.42)	12.37 (3.38)	15.61 (3.66)	15.09 (2.59)	6.73	33	.014
BPM RELAX	7.78 (1.62)	7.19 (1.03)	9.66 (6.75)	9.77 (2.65)	2.98	33	.094
BPM REC	11.96 (4.26)	11.26 (3.02)	12.06 (5.01)	13.15 (3.24)	8.77	33	.006

RSA respiratory sinus arrythmia, *PMR* progressive muscle relaxation, *DAPS-PTS-T* detailed assessment of posttraumatic stress-posttraumatic stress-total, *PCL* posttraumatic stress disorder checklist—civilian version, *BDI-II* beck depression inventory II, *ISI* insomnia severity index, *CRAVE* substance craving, scale range 1–10, *SDNN BASE* standard deviation of all normal-to-normal RR intervals at baseline, *SDNN RELAX* standard deviation of all normal-to-normal RR intervals at six-breaths-a-minute respiration training, *SDNN REC* standard deviation of all normal-to-normal RR intervals at recovery, *BPM* breaths per minute

[a] Degrees of freedom vary because of missing data

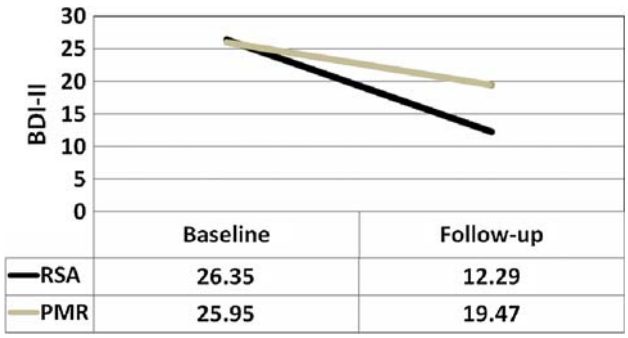

Fig. 1 Interaction Effects on the BDI-II from baseline to follow-up. *BDI-II* beck depression inventory, second edition

the PMR group. Because lower BPM can increase HRV, baseline and end of treatment BPM were added as a covariate to the HRV analyses. Results indicate that change in BPM, partially mediated the relationship between group and post treatment SDNN; end of treatment BPM, $F(3,30) = 3.08$, $p < .05$ and group assignment, $F(3,30) = 1.96$, $p < .1$.

To examine the relationship between changes in SDNN and changes in PTSD symptoms during the course of treatment, several hierarchical multiple regressions were performed with post-intervention PTSD score as the dependent variable, with baseline SDNN and PTSD scores entered in the first step and post-intervention SDNN scores entered in the second step. The same analyses were conducted with BDI scores as the dependent variable. Results for PCL-C total score revealed that post-intervention SDNN accounted for 17.8% of the variance in post-intervention PCL-C scores, incremental $F(1, 29) = 6.25$, $p = .019$. When examining the effects of change in SDNN on BDI scores, a statistical trend was found with post-intervention SDNN accounting for 6.2% of the variance in post-intervention BDI scores, incremental $F(1, 29) = 3.09$, $p = .09$. When BPM was added to the equation, change in SDNN remained a significant predictor of PCL-C changes, $t = 2.08$, $p < .05$. Change in BPM was not significantly associated with changes in PCL-C scores. There was no association between change in HRV and ISI scores or substance craving.

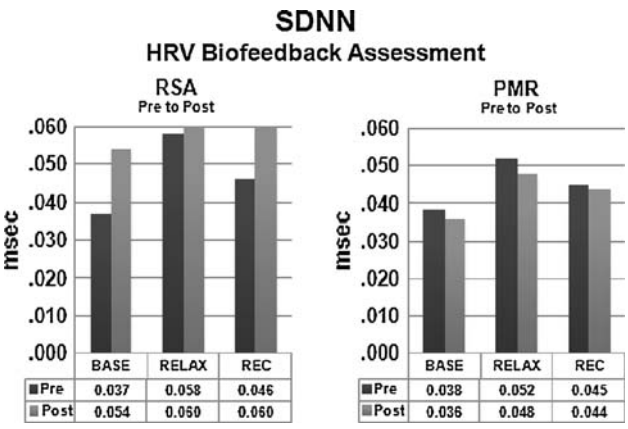

Fig. 2 SDNN from baseline to follow-up by group and assessment period. SDNN ≥ .050 ms is normal for healthy adults, *SDNN* standard deviation of all normal-to-normal RR intervals, BASE = 5 min: Baseline, RELAX = 10 min: Six-breaths-per-minute biofeedback training, REC = 5 min: Recovery

Dose-Response of the StressEraser and the PMR Exercise

The data from the StressEraser indicated that among group members there was a wide range of number of points achieved as well as efficiency. PMR group participants reported practicing the 20-min exercise an average of five to six times per week. However, the amount of practice for both groups was not significantly associated with any outcome measures.

Discussion

This controlled randomized pilot study is among the first to assess the feasibility of an RSA biofeedback treatment as an adjunctive intervention for traumatic stress and its related symptoms. The RSA group had significantly greater reductions in depression scores compared to PMR. Both groups significantly reduced PTSD symptom severity. Exploratory analyses indicated that, although not significant, the RSA group reduced substance craving from pre-intervention to post-intervention and both groups significantly reduced insomnia symptoms during the same period. HRV increased significantly more in the RSA biofeedback group than in the PMR condition from pre-intervention to post-intervention but this group difference was partially mediated by a significant reduction in resting respiration in the RSA group. Increases in HRV predicted concurrent PTSD symptom improvement even when controlling respiration rate (BPM). Overall, results from this study support further investigation of RSA biofeedback as an adjunctive treatment for persons with elevated PTSD symptoms.

This study adds to the body of research that links RSA biofeedback training with an increase in HRV indices and a reduction of psychiatric symptoms associated with trauma (Nishith et al. 2003). These findings are also consistent with previous research documenting a significant relationship between low baseline HRV and PTSD (Cohen et al. 1997, 1998, 2000). Both conditions had SDNN baseline means of less than the criterion indicated for healthy adults (Task Force 1996). After the 4-week intervention period, the baseline HRV for the RSA group was significantly higher as well as clinically within the normal range for

healthy adults, relative to the comparison group. Although this relationship was partially mediated by a decrease in resting respiration rate, the reduction in respiration is a notable finding in itself. The decrease indicates that participants may have learned to regulate their respiration rates in a relatively short time period. This acquisition of learning may point towards using reduced respiration as an additional psychophysiological intervention strategy for decreasing PTSD symptoms as recent research suggests that increased respiration rate is a predictor of the onset of PTSD (Bryant et al. 2008).

This study indicated that RSA biofeedback training might produce a decrease in symptoms associated with PTSD such as depression and insomnia. Karavidas et al. (2007) found similar reductions in depressive symptoms using HRV biofeedback. Previous intervention studies with major depressive disorder samples indicated that a decrease in depressive symptoms is associated with an increase in HRV (Balogh et al. 1993; Chambers and Allen 2002; Karavidas et al. 2007). Taken together, HRV and RSA biofeedback may be an effective emerging treatment for depressive disorders. In the current study, both interventions significantly reduced insomnia symptoms. Karavidas et al. (2007) also showed that HRV biofeedback decreased insomnia symptoms in a depressive sample. A significant decrease in both conditions is noteworthy because PMR is an empirically supported treatment for insomnia (Morin et al. 1999). Lastly, exploratory analyses indicated that only the RSA condition was associated with a trend towards reduced substance craving. This finding was in addition to the treatment participants were already receiving for SUDs, and should be explored more in future research.

Adding RSA biofeedback as an adjunct to empirically validated treatments for PTSD may result in better treatment outcomes. Higher vagal tone hypothetically may affect the diverse PTSD symptoms of both hyperarousal and avoidance (such as emotional numbing) because it regulates both hyperarousal and hypoarousal (Lehrer 2003). Empirically validated CBT modalities, including exposure therapy, for PTSD (Foa et al. 2000a) usually involve teaching patients relaxation skills to manage self-regulatory capacities, distress, and behavioral arousal (Frewen and Lanius 2006). Adjunctive skills training components may include muscle relaxation, imagery, and sensory awareness techniques, which alone are not empirically supported treatments for PTSD (Foa et al. 2000a). Adding RSA biofeedback to a skills training component may effectively assist patients in regulating arousal. Because there is no one "gold standard" treatment for PTSD, Foa et al. (2000b) urges clinicians to employ "creative integration of new approaches" (p. 540), which combine identified efficacious treatment interventions with experimental adjunctive ones.

Our study indicated that an increase in HRV was associated with a decrease in PTSD symptoms, even when controlling for resting respiration. Currently, elevated HR is the most salient identifying physiological feature of PTSD (Rabois et al. 2002). However, an HRV index may be more revealing physiological assessment of PTSD than HR activation. While measures of elevated HR dichotomously assess sympathetic arousal, HRV quantitatively measures the relative influence and interaction of both branches of the ANS on HR responsivity. Although the multi-site study by Keane et al. (1998) indicated that elevated HR was a primary, characterizing physiological feature of PTSD, the study also found that approximately 30% of PTSD subjects lacked HR reactivity to trauma cues. In addition, when HRV assessment studies ranked participants by their HRV amplitude they found that only those with the lowest indices had an accompanying elevated HR (Hopper et al. 2006) and a more prolonged HR arousal (Sack et al. 2003). Noting similar physiological responsivity differentials in other studies, several researchers have hypothesized that PTSD may have two distinct physiopathological processes, one being an arousal subtype and the second a dissociative one (Bremner 1999; Foa et al. 1995; Lanius et al. 2006). Future research might benefit from assessing additional HRV frequency variables (in addition to SDNN, a time domain one), which may clarify how the two ANS branches may discretely influence HR activation and physiologically define the hypothetical PTSD subtypes.

There were several limitations to the study. This study had a small sample size and the results cannot lead to definitive conclusions. The interventions were conducted in conjunction with a residential treatment program; it is unclear how the program influenced trauma symptoms, and whether the overall symptom improvement found in both groups may be due in fact to the treatment program itself. Moreover, while PTSD and SUDs are highly comorbid (Ouimette et al. 2002), the effectiveness of treatment strategies is unknown for the concurrent disorders (Oiumette et al. 2003). A methodological limitation was that the treatment protocol was not standardized or supervised, and data indicated mixed treatment adherence. For the PMR group, the daily practice amount or time cannot be objectively verified while the use of the StressEraser was heterogeneous. Thus, it remains unclear how much RSA biofeedback training is necessary to affect vagal mechanisms because no differences between high and low users of the StressEraser were found on any measure. It is possible that minimal practice can have therapeutic effects. Alternatively, other factors such as the novelty of the RSA intervention could have created significant non-specific effects. Another limitation is that several variables known to affect HRV indices, including caffeine intake and daily exercise, were not evaluated, and it is unknown whether the

clinical and statistical differences could be further explained by them. It is also unclear why no dose-response was found for the RSA intervention. Finally, results from this specialized group and setting may not necessarily generalize to other samples.

RSA biofeedback may be a viable treatment intervention for depression as well as a possible one for PTSD, insomnia, and SUDs. Continued research utilizing RSA biofeedback as a treatment intervention either with the StressEraser or with the standardized office- based HRV biofeedback training protocol, or a combination of both, is recommended. Future studies may further clarify the interrelationship between autonomic functioning and PTSD and its associated features.

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